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1. Behrman RE, Kliegman RM, Arvin AM, eds. Nelson textbook of pediatrics. 15th ed. Philadelphia: W.B. Saunders, 1996.

2. Rudolph AM, ed. Rudolph's pediatrics. 20th ed. Stamford, Conn.: Appleton & Lange, 1996.

3. Emerging and communicable diseases: surveillance and control. Geneva: World Health Organization, 1997.

4. Henrickson KJ, Hoover S, Kehl KS, Hua W. National disease burden of respiratory viruses detected in children by polymerase chain reaction. *Pediatr Infect Dis J* 2004;23:Suppl:S11-S18.

Monkeypox in the Western Hemisphere

TO THE EDITOR: Infection control was a major issue for investigators attempting to minimize the emergence of monkeypox in the United States, as reported by Reed et al. (Jan. 22 issue).¹ On June 7, 2003, three Illinois residents with a febrile rash syndrome presented to a community hospital. Hospital staff reported the cases that evening to the Illinois Department of Public Health, which recommended diagnostic testing, collection of contact information, and admission under contact and airborne precautions.

Infection control was efficiently implemented, despite the absence of preexisting policies specific to this pathogen and uncertainty regarding best practices for the prevention of person-to-person transmission.² The hospital's participation in the Top Officials 2 (TOPOFF 2) bioterrorism exercise in May 2003,³ smallpox training activities, and past management of an imported case of Lassa fever⁴ enhanced the execution of infection-control protocols.

This outbreak tested a hospital's preparedness to respond to an unusual communicable agent. Had the outbreak been larger, the hospital's isolation facilities would have been insufficient. Hospitals should critically evaluate their capacity to implement rapid syndrome-based isolation precautions for emerging disease outbreaks.⁵

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1. Reed KD, Melski JW, Graham MB, et al. The detection of monkeypox in humans in the Western hemisphere. *N Engl J Med* 2004; 350:342-50.

2. Jezek Z, Grab B, Dixon H. Stochastic model for interhuman spread of monkeypox. *Am J Epidemiol* 1987;126:1082-92.

3. Top Officials (TOPOFF) exercise series: TOPOFF 2: after action summary report for public release. Washington, D.C.: Department of Homeland Security, December 19, 2003. (Accessed April 1, 2004, at http://www.dhs.gov/interweb/assetlibrary/T2_Report_Final_Public.doc.)

4. Holmes GP, McCormick JB, Trock SC, et al. Lassa fever in the United States: investigation of a case and new guidelines for management. *N Engl J Med* 1990;323:1120-3.

5. Bioterrorism readiness plan: a template for healthcare facilities. Washington, D.C.: Association for Professionals in Infection Control and Epidemiology, Centers for Disease Control and Prevention, April 13, 1999. (Accessed April 1, 2004, at <http://www.apic.org/educ/readinow.cfm>.)

TO THE EDITOR: In their report on the U.S. monkeypox outbreak (72 cases), Reed et al. cite African outbreaks of 23 and 88 cases. By doing so, the authors risk minimizing the magnitude of the problem in Africa, where the disease has been endemic since the 1970s, with multiple outbreaks, including one outbreak of 419 cases in 1996–1997.¹ The large size of this African outbreak may have resulted from increased contact with animals in a population of persons displaced by civil war.² High rates of human exposure to monkeypox may occur in other scenarios, such as the infection of wild rodents in U.S. cities.

The animal reservoir for human monkeypox remains unknown.³ Although prairie dogs are the probable source of transmission in most U.S. cases, there has been human transmission from other species. A rabbit (*Leporidae* family) that was exposed to a diseased prairie dog was implicated as the source of human infection in at least one U.S. case.⁴

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1. Human monkeypox — Kasai Oriental, Democratic Republic of Congo, February 1996–October 1997. *MMWR Morb Mortal Wkly Rep* 1997;46:1168-71.

2. Heymann DL, Szczeniowski M, Esteves K. Re-emergence of monkeypox in Africa: a review of the past six years. *Br Med Bull* 1998;54:693-702.
3. Di Giulio DB, Eckburg PB. Human monkeypox: an emerging zoonosis. *Lancet Infect Dis* 2004;4:15-25.
4. Multistate outbreak of monkeypox — Illinois, Indiana, and Wisconsin, 2003. *MMWR Morb Mortal Wkly Rep* 2003;52:537-40.

THE AUTHORS REPLY: Drs. DiGiulio and Eckburg express concern about the ongoing, significant disease burden associated with human and animal monkeypox infections in Africa. We share their concern. In our article, we cited two monkeypox epidemics in the Democratic Republic of Congo that were similar in size to the U.S. outbreak.^{1,2} We provided this information to place the scope of the current investigation in perspective, but with no intention of minimizing perceptions of the impact monkeypox is having in Africa. In fact, the many epidemics that have occurred in Africa should serve as a sobering reminder of the significant problem monkeypox could become in North America if enzootic transmission cycles of infection became established in our native wildlife species.

Investigation of the animal reservoir (or reservoirs) of monkeypox virus associated with the U.S. outbreak is ongoing. Drs. DiGiulio and Eckburg refer to an early report linking a suspected case in a human with a rabbit that became ill after exposure to an ill prairie dog at a veterinary clinic.³ Although rabbits are known to be susceptible to experimental infection with monkeypox virus, subsequent tests in the animal and the patient, according to that report, were negative. We are not aware of any documented cases of illness that resulted from transmission of monkeypox virus from rabbits to humans in the United States.

We agree with Dr. Huhn and colleagues that it is

important for medical facilities to evaluate critically their capacity to implement isolation precautions for emerging infectious disease outbreaks rapidly. In this outbreak, the Internet and other forms of communication technology played a critical role in piecing the puzzle together and enhancing the public health response.⁴ It is fortunate that the U.S. monkeypox outbreak was limited to 72 confirmed or probable human cases, none of which were fatal. The outbreak was contained by rapid identification of the etiologic agent and reservoir and by intense, multidisciplinary, highly collaborative efforts to control the outbreak and prevent new cases from occurring. Many more cases could well have occurred had there not been aggressive deployment of local, state, and federal resources to limit the outbreak.

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1. Hutin YJF, Williams RJ, Malfait P, et al. Outbreak of human monkeypox, Democratic Republic of Congo, 1996 to 1997. *Emerg Infect Dis* 2001;7:434-8.
2. Meyer H, Perrichot M, Stemmler M, et al. Outbreaks of disease suspected of being due to human monkeypox virus infection in the Democratic Republic of Congo in 2001. *J Clin Microbiol* 2002;40:2919-21.
3. Multistate outbreak of monkeypox — Illinois, Indiana, and Wisconsin, 2003. *MMWR Morb Mortal Wkly Rep* 2003;52:537-40.
4. Reed KD. Monkeypox, Marshfield Clinic and the Internet: leveraging information technology for public health. *Clin Med Res* 2004;2:1-3.

Off-Pump versus On-Pump Coronary Bypass Surgery

TO THE EDITOR: In the article by Khan et al. (Jan. 1 issue),¹ the conclusion regarding the inferiority of graft patency in patients who have undergone off-pump bypass surgery is premature. Before initiation of the study, each of the two participating surgeons had performed, on average, only 49 off-pump graft procedures, and they performed a total of only 75 such procedures (in 27 patients) during the trial. The study identified disparate reductions in overall patency for grafts in the right-coronary-artery dis-

tribution and for those involving radial-artery conduits, as compared with previously published results.² There is a direct correlation between the number of procedures performed by a surgeon and the clinical outcome,^{3,4} suggesting that patency rates are a function of surgical experience. In addition, the authors did not use apical suction devices or routine intracoronary shunting, both of which facilitate technical precision in the creation of an anastomosis, particularly in difficult-to-graft terri-